General Session 2: Decision Making in CHB Patients Management

Saturday, July 16, 2011, 15:30–17:00
Meeting Room 309

GS2.1 Indication to start therapy – possible, optional and mandatory

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Abstract not available

GS2.2 Treatment endpoints for HBV

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Chronic hepatitis B virus (HBV) infection is still a major health problem worldwide. In the past decade, much progress has been made in the understanding and management of chronic HBV infection and related liver diseases. Most importantly, the introduction of universal vaccination has significantly reduced the incidence of perinatal new HBV infection in most countries. In the past decade, there have also been great advances in the development of anti-viral therapy. Ideally, therapies would be able to prevent adverse clinical outcomes such as the development of cirrhosis, end-stage liver disease and hepatocellular carcinoma. However, these clinical endpoints typically take decades to occur and are therefore impractical targets for clinical practice. As a result, certain surrogate biomarkers that correlate with long-term outcomes are frequently used to evaluate efficacy of anti-viral therapy. Of the serological and virological endpoints that have been used, none has been shown to be ideal. Suppression of viral replication, as measured by serum HBV DNA levels, has been a major goal of therapy. Although useful, the significance of viral levels depends on the stage of disease, degree of liver damage, and the type of therapy. Besides, rebound of serum HBV DNA may occur in a large proportion of patients receiving oral antiviral agent. Hepatitis B e antigen (HBeAg) seroconversion is still an important treatment endpoint in HBeAg-positive patients. However, the durability of nucleos(t)ide analogue treatment-related HBeAg seroconversion is not satisfactory; and the emergence of HBeAg-negative disease is another concern. Loss of hepatitis B surface antigen (HBsAg) is considered close to the cure of the disease and is associated with improved clinical outcomes; nevertheless it is rarely achieved with current anti-vial therapies. Finally, liver biopsy, traditionally considered as the gold standard, is invasive, prone to sampling error, and not accepted by many patients. In short summary, there is no ideal biomarker for the evaluation of therapy for chronic hepatitis B. Patients should be carefully monitored after stopping anti-viral drugs. Challenges remain in the development of criteria for treatment cessation and best treatment endpoints. HBsAg quantitation is now being actively investigated for its role in determining the natural history of CHB and whether the on-treatment kinetics could predict response to anti-viral therapy. The benefit and limitations of using these endpoints will be presented in this meeting.

GS2.3 Management of CHB patients: Considerations for the difficult cases and special populations

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Abstract not available

Concurrent Session 14: Viral Hepatitis and Liver Cancer

Sunday, July 16, 2011, 09:15–10:45
Meeting Room 311A

CS14.1 The risk of hepatocellular carcinoma in HCV-related chronic hepatitis

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For the last 20 years, we are trying to elucidate the natural courses of HCV infections from acute hepatitis to the development of Hepatocellular Carcinoma. We have followed 3000 patients almost 15 years, and it was found that the progression of fibrosis is a key component to develop hepatocellular Carcinoma.
Furthermore, the eradication of HCV virus in fact regress the extend fibrosis and eventually reduce the cancer risk.
Recent molecular diagnostic analysis revealed certain SNPs may predict the outcome of the development of Hepatocellular Carcinoma by GWAS (Genome Wide Association Study). The genetic analysis may help us to identify the individuals to the development Hepatocellular Carcinoma more precisely in the future.
These strategies, along with the improvement of treatments by themselves, are actually ending up now to the gradual reduction of the Hepatocellular Carcinoma death in Japan.

CS14.2 Risk assessment of HBV-related HCC development using liver stiffness measurement

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Background and Aims: Liver stiffness measurement (LSM) using FibroScan accurately assesses the degree of liver fibrosis and the risk of hepatocellular carcinoma (HCC) development in patients with chronic hepatitis C. This study investigated the usefulness of LSM as a predictor of HCC development in patients with chronic hepatitis B (CHB).
Methods: A total of 1,130 patients with non-biopsy-proven CHB who underwent LSM between May 2005 and December 2007 were enrolled in this prospective study. After LSM was performed, patients attended regular follow-up as part of a surveillance program for the detection of HCC.
Results: The mean age of the patients (767 men, 363 women) was 50.2 years, and the median LSM was 7.7 kPa. Six hundred seventy-two (59.5%) patients received antiviral treatment before or after enrollment. During the follow-up period (median, 30.7 months; range, 24.0–50.9 months), HCC developed in 57 patients (2.0% per 1 person-year). The 1-, 2-, and 3-year cumulative incidence rates of HCC were 0.80%, 3.26%, and 5.98%, respectively. On multivariate analysis, together with old age, male sex, heavy alcohol consumption (>80 g/day), serum albumin, and hepatitis B e antigen positivity, patients with a higher LSM (>8 kPa) were at a significantly greater risk of HCC development, with the following hazard ratios: 3.07 (95% confidence interval [CI], 1.01–9.31; $P = 0.047$) for LSM 8.1–13 kPa; 4.68 (95% CI, 1.40–15.64; $P = 0.012$) for LSM 13.1–18 kPa; 5.55